IS A Q WAVE INFARCTION NECESSARY FOR THE GENERATION OF A LATE POTENTIAL ?

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The signal averaged electrocardiogram (SAE) is predictive of future arrhythmic events after a myocardial infarction (MI). However, it is unknown whether the type of MI, Q wave vs non-Q wave, affects the generation of potential substrate for malignant ventricular arrhythmias.

SAE was performed on 50 consecutive patients within one week of an enzymatically documented MI. Patients with prior MI or bundle branch block were excluded. Late Potentials (LP), defined by an RMS40 ≤ 25uV, were present in 13 (26%) of these patients . Ejection fractions in patients with and without LP's were not significantly different (44±16 vs 49±17% respectively, p=NS).

MI	N	LP Frequency*	EF%†
non-Q	22	9%	51%
Q	28	39%	45%
		*p=0.04	†p=NS

In summary, non-Q wave infarctions are notable for the paucity of Late Potentials. This lack of an arrhythmogenic substrate as manifest by the signal averaged electrocardiogram suggests that the early mortality in this population may be primarily on the basis of reinfarction and not ventricular arrhythmias.

THE PRESENCE OF LATE POTENTIALS ON THE SIGNAL AVERAGED ELECTROCARDIOGRAM IN PATIENTS WITH MYOTONIC DYSTROPHY.

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Myotonic dystrophy is associated with sudden death and is ascribed to the development of advanced heart block. The presence of late potentials (LP) is predictive of arrhythmic events in certain populations. We evaluated for the presence of LP on the signal-averaged ECG(SA-ECG) (40Hz filter) in 25 myotonic dystrophy patients(pts) (15 males, age=41±11yrs) with no history of cardiac symptoms. LP criteria (time domain analysis) included a filtered QRS >114ms, a terminal RMS voltage <20uV or a terminal low amplitude signal (<40uV) >38ms. One subject was excluded (bundle branch block). 75% met 1 criteria, 67% met 2 criteria, and 29% met all 3 criteria for LP. Using both time and frequency data to produce a spectro-temporal map of the SA-ECG(x lead), an abnormal contour consistent with late potentials was demonstrated in 20 of 22 subjects tested. A prolonged PR (208 vs 178ms, p<.04) and QRS (109 vs 92ms, p<.07) interval were noted in those subjects with time domain analysis criteria for late potentials. A linear correlation was noted between the PR interval and each LP criteria, especially the filtered QRS duration (r=.652, p<.001). The presence of LP did not correlate with age (40.4 vs 40.3 yrs), sex, extent of neurologic disease, or with a family history of arrhythmic events.

In conclusion, late potentials are commonly seen in pts with myotonic dystrophy. This finding raises the possibility that ventricular arrhythmias may contribute to the known risk of sudden death in this group.

LATE POTENTIALS IN PATIENTS WITH MITRAL VALVE PROLAPSE WITHOUT VENTRICULAR TACHYCARDIA.

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To determine the incidence and significance of late potentials (LP) in patients with mitral valve prolapse (MVP), we performed signal-averaged electrocardiography and ambulatory holter monitoring on 39 patients with moderate to severe MVP on physical examination and two dimensional echocardiography. LP were present if RMS-voltage (RMS) in terminal 40ms of 40Hz filtered vector magnitude QRS <25 uV or low amplitude signal (LAS) <40 uV exceeded 39ms. We identified 12 patients (31%) with LP in Group I. Group II consisted of the remaining 27 subjects. All patients were followed for a mean of 34±12 months. No deaths occurred.

	Group I	Group II	
RMS (uV)	17.0±6.8	57.9±4.6	p<.009
LAS (ms)	43.0±8.2	25.3±6.6	p<.0001
Duration of QRS (ms)	100.0±8.3	90.1±9.3	p<.008

Comparing Groups I and II, there were no significant differences in clinical characteristics, supraventricular or ventricular arrhythmias, or long term prognosis. We conclude there is a high incidence of LP in asymptomatic MVP patients, and this seems to be a benign finding in contrast to patients with other cardiac diseases.

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Author Present: 2:00PM–3:00PM
Hall C, New Orleans Convention Center
Antiarrhythmic Drugs

TORSADE DE POINTES LATE AFTER I.V. QUINIDINE IN CONSCIOUS DOGS WITH AV-BLOCK. COMPARISON WITH ACUTE EFFECTS.

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In-vitro quinidine (Qd) effect on cellular repolarization has recently been shown to be delayed, with action potential still increasing beyond 3h of exposure. Thus acute in-vivo experiments could underestimate QT-dependant arrhythmogenesis of Qd.

We studied acute and late Qd-induced ventricular arrhythmias (VA) on conscious dogs with AV-block and diuretic-induced hypokalemia (hK). In 8 control dogs (ventricular cycle: RR= 1600±120 ms) with hK (2.6-2.9 mmol/l), holter ECG showed only rare ventricular ectopic beats, isolated (VPB:n=1) or repetitive (VPB:n=3). Nine dogs in hK (2.6-3.2 mmol/l) were tested with i.v. Qd (10 mg/kg in 10 min followed by 30 µg/kg/min for 60 min) and were evaluated for acute effects (1h ECG) and delayed effects (24h Holter). At 30 min quinidine plasma level was 3.0±0.4 mg/l, RR remained unchanged (1580±170 ms), but QT increased from 317±11 to 345±12 ms (p<0.01). Acute VA were limited to runs of polymorphic or monomorphic ventricular tachycardia (VT:n=4), VPB's (n=3) or VPB's (n=1). Subsequent 24h ECG demonstrated neither RR (1520±60 ms) nor QT (365±8 ms) variations but showed delayed VA beyond 3 hours in 6 dogs: VPB (n=3), VT (n=1) and typical 'torsade de pointes' (TdP) for 2 dogs, one of them resulting in ventricular fibrillation and sudden death.

These late TdP could be related to delayed effect of quinidine on QT at slow heart rate and/or regression of autonomic changes due to i.v. Qd.